Bandolier 141

Independent evidence-based thinking about health care

On bad things

Bandolier this month is all about bad things happening: the bad things that happen when you smoke cigarettes, when you take "recreational" drugs and drive, to your bowel when you take NSAIDs, and bad things possibly related to acne treatment. Bad things happen a lot. While in the long run we are all dead, we still strive valiantly to avoid bad things, so we might do more to understand them.

Dose response

One thing often forgotten about adverse events, particularly but not only with drugs, is that they are usually related to dose. The more you take the worse the event or the more likely it is to occur. Not a new thought, this. "*All drugs are poisons, the benefit depends on the dosage*" (Philippus Theophrastrus Bombast of Aureolus Paracelsus), which is why "*the best doctor gives the least medicines*" (Benjamin Franklin). Cigarette smoking and driving on drugs provide two nice examples, and good reasons for eschewing both.

Seek and ye shall find, or not

Another oft forgotten truism for adverse events is that the more you look, the more you find. Aspirin and NSAIDs have been with us for decades. We thought we knew them. Yet come along with proper modern studies and we find what we chose to ignore previously. As well as causing havoc in the stomach and duodenum, much of the rest of the bowel is at risk of damage as well. Rates of damage are high, and what might protect in the stomach does not work further down.

However hard you look, though, for some very rare adverse events you find nothing substantive, only ephemera. Conclusive links between acne treatments and suicide have not been found. But the evidence we have is that where there is history, personal or family, of mental problems, acne treatments can be dangerous. Not what treatment, but who to treat.

In this issue

Cigarette smoking

Dose-response

In Bandolier's infancy it looked at some of the facts about the negative health effects of smoking (Bandolier 7), particularly the strong dose-response for lung cancer. Now we have smoking bans, fewer people smoke, and more of those who do smoke, smoke less.

With the changes of the last decade, it is worth revisiting the issue of dose response, particularly when we have good evidence at the lower end of the dose-response curve [1], and for effects other than lung cancer.

Dose-response study

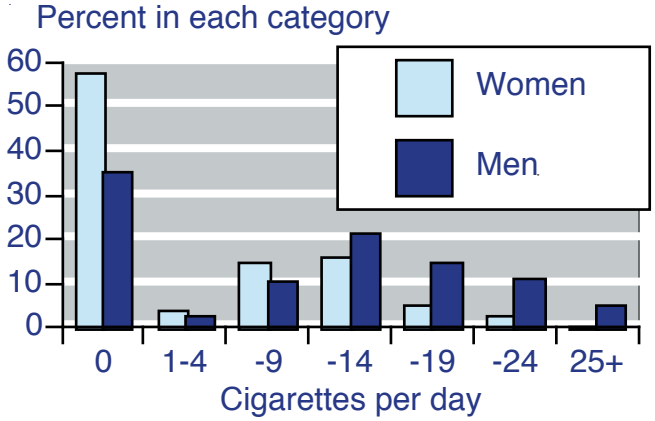
Inevitably the evidence comes from an observational study. In this case [1] it stems from screening examinations for cardiovascular disease in parts of urban and rural Norway that began in the 1970s. The screening included questionnaires about cardiovascular disease, demographic measures, blood tests and questions about smoking habits.

Excluded were people with a history of chronic disorders like heart disease, diabetes, or hypertension, and ex-smokers or pipe smokers. That left about 19,000 women and 24,000 men for whom death and cause of death could be determined by the end of 2002.

Results

In this sample, 42% of women and 65% of men smoked. Men who smoked mostly smoked 10 cigarettes a day or more, while women rarely smoked more than 15 cigarettes per day. The lowest consumption, 1-4 cigarettes per day, involved 4% of women and 3% of men (Figure 1).

Figure 1: Smoking habits of 23,500 men and 19,000 women in Norway in the mid 1970s

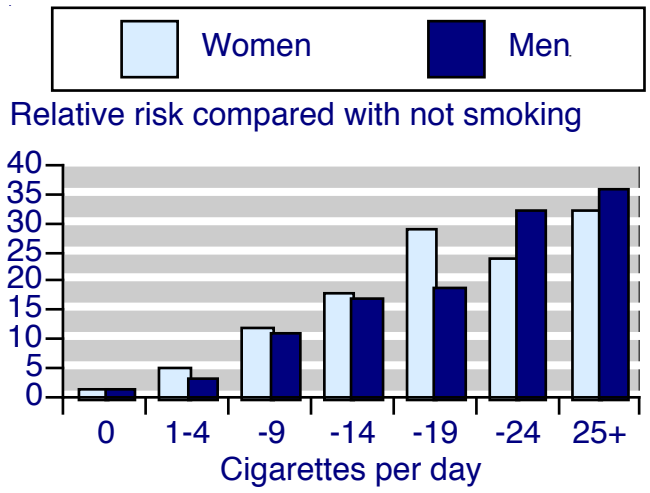


Across the whole population, the average age at the screening event was about 42 years, and BMI about 24. In women who smoked, the duration of smoking averaged 12 years for the lightest smokers (1-4 cigarettes a day) to 20 years in the heaviest (25+ a day). In men who smoked, duration ranged from 18 to 24 years. Average total cholesterol was quite high, over 6.5 mmol/L.

Figures 2 to 5 show the actual number of events per 100,000 person years for men and women, for all cancer, for lung cancer, for ischaemic heart disease, and for all death. There was a strong dose response with cigarette consumption, with more cancers, lung cancers, cases of ischaemic heart disease, and death with more cigarettes smoked. Ischaemic heart disease affected women less than men (Figure 4).

The strongest dose response was for lung cancer (Figures 3 and 6), where smoking 10-15 cigarettes a day increased the risk by 17-fold, and 25 a day or more by about 35-fold compared with not smoking. For all-cause deaths, all cancers, and ischaemic heart disease there was higher relative risk with more cigarettes. Even with 1-4 cigarettes a day there was a significantly increased risk of all cause mortality, ischaemic heart disease, and lung cancer in both men and women. Relative risks were about 1.5 for all cause mortality, 2.8 for ischaemic heart disease, and 3-5 for lung cancer.

Figure 6: Relative risk for lung cancer according to cigarette consumption



Comment

No apologies for a revisit. In the UK parents can still take children into pubs for a meal, into atmospheres with a high proportion of tobacco smoke. Smoking bans in many parts of the world have been successfully implemented, and it is only when you go somewhere without one that one realises just how much of an imposition smoking can be.

Getting down the dose response curve is good if you are at the top. Stopping people being on the curve at all is so much better.

Reference:
1 K Bjartveit, A Tverdal. Health consequences of smoking 1-4 cigarettes per day. Tobacco Control 2005 14: 315-320.

Figure 2: All cancer by cigarette consumption

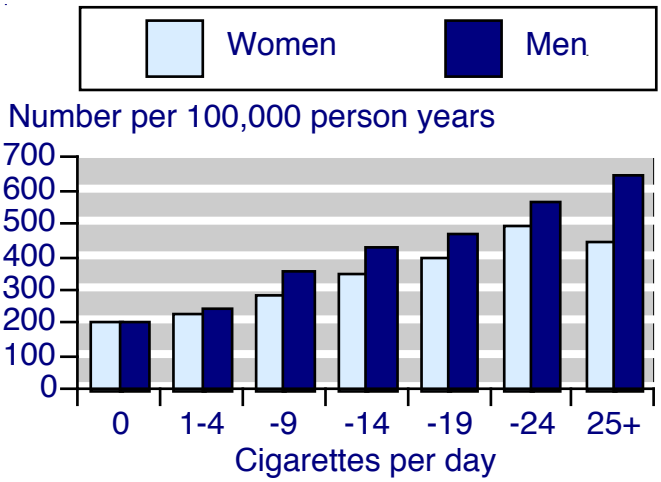


Figure 3: Lung cancer by cigarette consumption

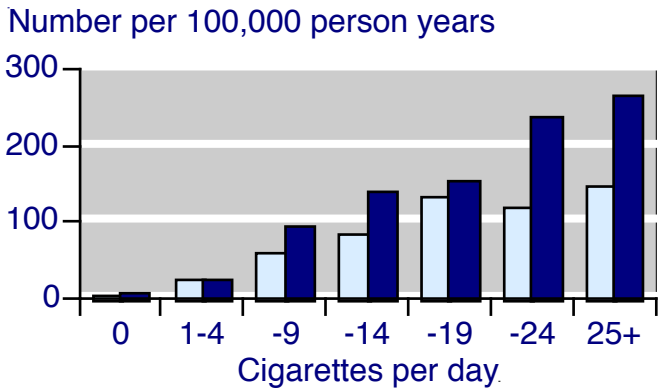


Figure 4: Ischaemic heart disease by cigarette consumption

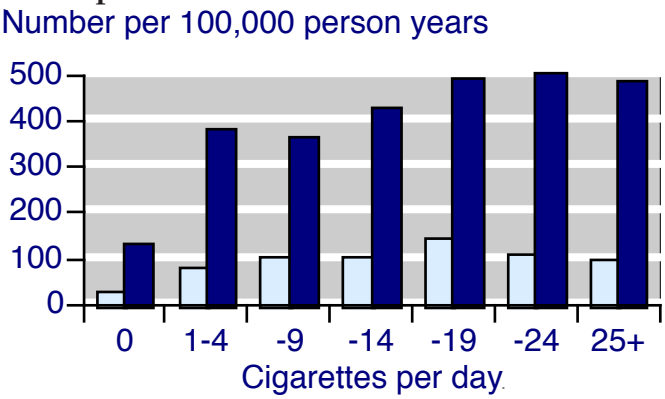
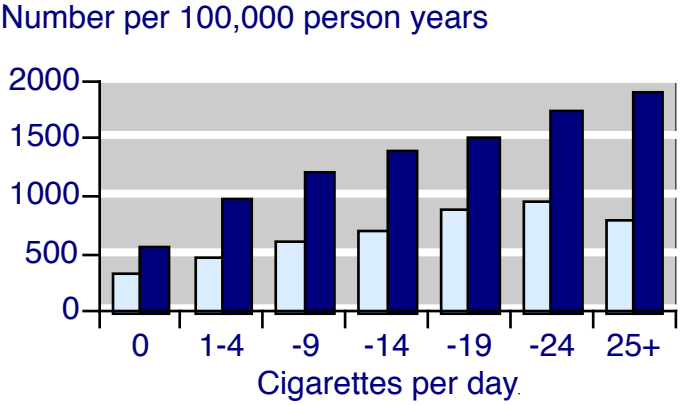


Figure 5: All-cause mortality by cigarette consumption



Drugs and driving

We know that people who drink alcohol and have blood alcohol concentrations above a certain level are much more likely to have a motor vehicle accident than those who have no alcohol present. It is why we have strict drink-driving regulations. But what about drugs, particularly the so-called recreational drugs? This is something of a moving target because patterns of recreational drug use change over time. A quick review of some recent studies from around the world tells that drugs and driving is at least as big a problem as alcohol and driving.

USA [1]

The setting was a cohort of traffic accident victims admitted during a six-month period to a trauma centre servicing most of Maryland, and for whom there was both a blood alcohol result and a sufficient urine specimen for drug analysis. Demographic variables were recorded, and toxicology results that could have resulted from medications administered during treatment were disregarded. Status (driver, passenger, pedestrian) was not available.

Results

Over the period there were 322 eligible patients, predominantly (71%) male, and about half were aged under 35 years. Overall 59% had a positive toxicology test, and drugs, with and without alcohol, were involved in almost three-quarters of the positive results (Figure 1).

Figure 1: Toxicology tests in RTA victims in Maryland

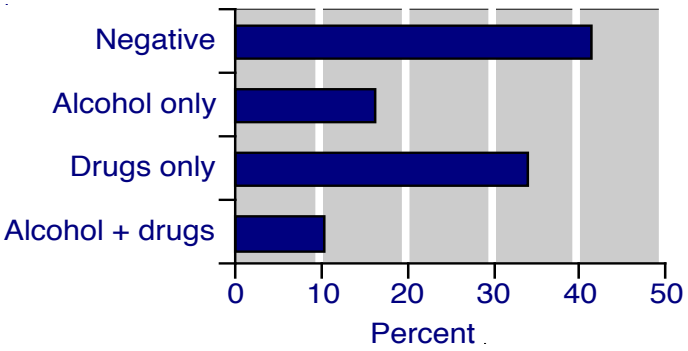
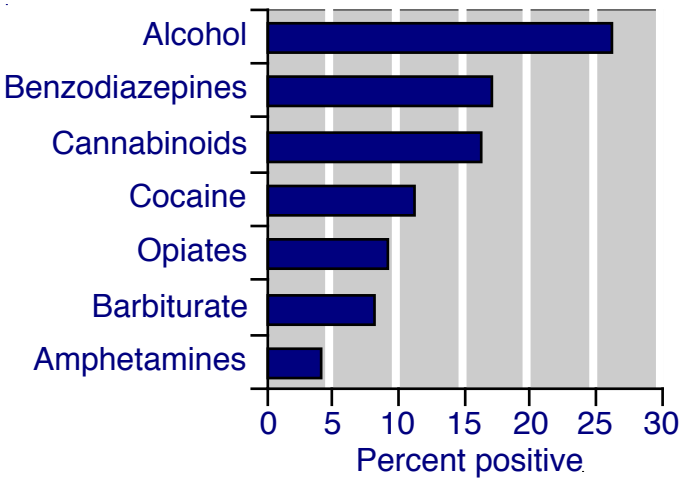


Figure 2: Ranking of positive toxicology tests in Maryland



Blood alcohol levels were above the drink drive limit of 80 mg/dL in all but three of the positives, and the average alcohol concentration in those with alcohol present was 210 mg/dL. Alcohol was the commonest single drug present, with benzodiazepines and cannabinoids also present in over 15% (Figure 2). Positive tests were found less frequently in women than men, except for benzodiazepines.

Australia [2]

Here the setting was 3,398 fatally injured drivers from three Australian states between 1990 and 1999. Only on-road crashes were included, and any considered to be suicide were excluded. Drivers were identified mainly from coroners' records and central toxicology laboratories. Analysis of each case analysed the degree of responsibility of each driver for the accident, an index of culpability divided into responsible for the crash, contributor, or not responsible.

Results

Most were car drivers (77%) or motorcyclists (19%). Most (about 80%) were men, and the average age was about 35 years, with a range of 12 to 92 years. Alcohol above 50 mg/dL in blood was found in 29%, drugs of any type 27%, and 10% of cases involved alcohol and drugs. The most commonly found drugs were cannabinoids (14%), opioids (5%), stimulants (4%) and benzodiazepines (4%).

Using those drivers who tested negative for alcohol or drugs as controls, drivers in the age range 30-60 years were less likely to be responsible for the accident. The ratio of drivers responsible to those not responsible was much higher (11) for positive cases than negative cases (3). High ratios were found for high blood alcohol (34), single vehicle crashes (27), drivers aged 17 years or below (12), and positive tests for cannabinoids (10) and psychoactive drugs (11) only.

Responsibility for accidents appeared to be concentration-related, at least for alcohol and cannabinoids. For alcohol (Figure 3), compared with those drivers without alcohol or drugs, the odds ratio for being responsible for the crash increased with blood alcohol concentration. Higher cannabinoid concentrations also raised the likelihood of driver responsibility.

Figure 3: Driver responsibility for crash, odds ratio by blood alcohol compared with no alcohol detected

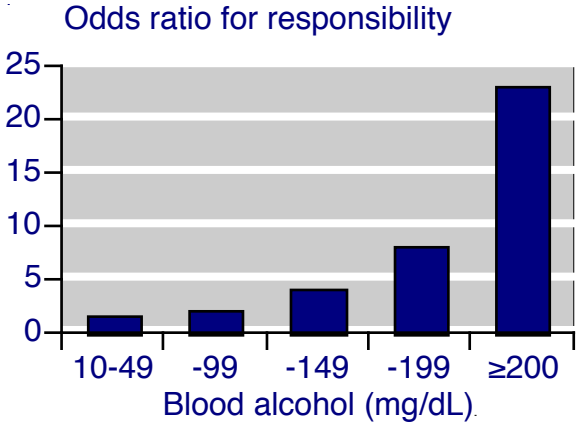
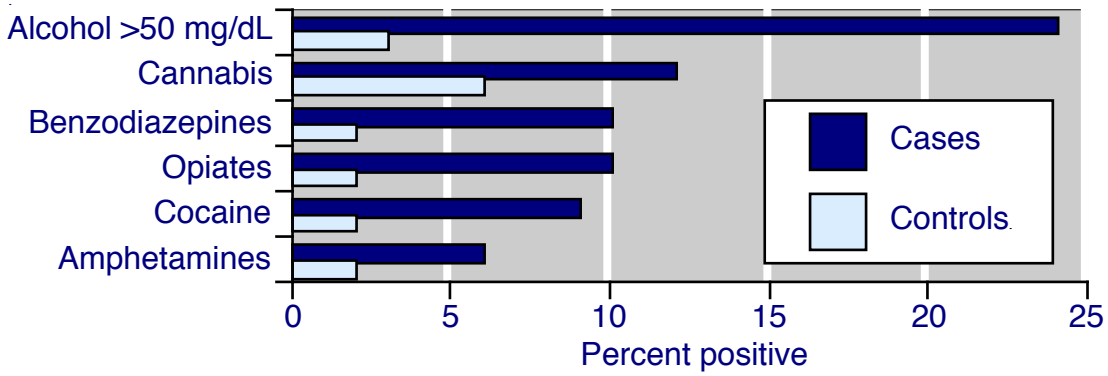


Figure 4: Positive toxicology tests in Dutch drivers in accidents involving injury, compared with randomly selected non-crash drivers



Holland [3]

A different approach was taken for controls in Holland. Here cases were car or van drivers involved in road crashes and needing hospital treatment in 2000 and 2001, in a population of 350,000. Controls were drivers recruited at random while driving on public roads. As best as possible, controls were drivers matched for age and sex, and for time of day. There were 110 cases and 816 controls.

Results

Three quarters of the cases were men, with an average age of 39 years. Controls were generally similar in demographics. Alcohol or drug tests were positive in 40% of cases, compared with 14% of controls. Alcohol was commonest, followed by cannabis and benzodiazepines (Figure 4). Only the use of a single drug without alcohol had a similar incidence (9%) in both cases and controls. Compared with no drug, use of multiple drugs plus alcohol carried a 100-fold increased risk of road accident injury.

New Zealand [4]

Drivers involved in 571 car crashes involving death or hospital admission of at least one occupant were compared for cannabis use with 588 randomly selected drivers in and around Auckland, with a population of about 1.1 million, in 1998 and 1999.

Results

The average age of crash drivers was 37 years, and 59% were men. Drivers and controls were well matched. Blood alcohol concentrations above 50 mg/dL were found in 23% of crash drivers and 1% of controls. Cannabis use within the last three hours was admitted by 6% of drivers involved in crashes, and 10% admitted using cannabis once a week or more. The figures for controls were below 1%.

Comment

These four studies come from different parts of the world, at different times over the past decade or so. Yet they demonstrate remarkable consistency. Alcohol remains the major factor, but with cannabinoids and benzodiazepines not that for behind in most of the studies.

Where dose was examined, there was a significant dose response for alcohol and cannabinoids. In the large Australian study, cannabinoid concentrations ranged from less than 1 μg/L to a staggering 228 μg/L, with the median value of 9 μg/L. Concentrations above 5 μg/L have been particularly associated with responsibility for accidents. In the US study, mean alcohol concentrations were 2-4 times the legal limit. For both alcohol and cannabis, where it was present it was present in concentrations known to cause problems.

Cannabis was found consistently in accident victims or drivers. It is not an easy drug to understand, with problems with acute and habitual use. A comprehensive, if not overtly systematic, review [5] pulls together evidence from various sources for those wanting a more detailed appreciation. The bottom line from the review is that cannabis makes it more difficult to drive on simulators, is associated with car accidents, and that alcohol and cannabis combined is an explosive mix that produces severe impairment of cognitive, psychomotor, and actual driving performance.

On a technical note, these four studies each choose different populations for cases and controls. The cases can be drivers or any person involved in an accident; they can be dead or alive. Controls were those testing negative, or randomly chosen drivers. In the latter case, a significant minority refused to cooperate - with implications for how many people take drugs and drive. It could be substantially more than these surveys revealed.

References:

1 JM Walsh et al. Epidemiology of alcohol and other drug use among motor vehicle crash victims admitted to a trauma center. Traffic Injury Prevention 2004 5: 254-260.
2 OH Drummer et al. The involvement of drugs in drivers of motor vehicles killed in Australian road traffic crashes. Accident Analysis and Prevention 2004 36: 239-248.
3 KL Movig et al. Psychoactive substance use and the risk of motor vehicle accident. Accident Analysis and Prevention 2004 36: 631-636.
4 S Blows et al. Marijuana use and car crash injury. Addiction 2005 100: 605-611.
5 JG Ramaekers et al. Dose related risk of motor vehicle crashes after cannabis use. Drug and Alcohol Dependence 2004 73: 109-119.

NSAIDs and bowel injury

The trouble with NSAIDs is that, despite their effectiveness for many types of acute and chronic pain, we continue to discover that they are injurious, with many different adverse effects. As well as being involved in renal failure, congestive heart failure, and bleeding in stomach and duodenum, there is now an unfolding story about additional bowel injury.

In the 1990s the use of radioactive indium-labelled white cells showed that faecal excretion of white cells, a marker of intestinal inflammation, was elevated with oral NSAID. Calprotectin, a calcium binding protein found in neutrophilic granulocytes, monocytes, and macrophages, which resists faecal degradation can also be used as a marker. Use of the test in 312 patients taking NSAIDs showed that 44% had raised faecal calprotectin concentrations, much the same as estimates with indium studies [1].

Capsule endoscopy is a technique that allows direct visualisation of the bowel, with the pictures captured on video. Injury to the bowel can be seen directly. Three new studies have used the technique to study bowel injury with NSAIDs.

Prevalence study with NSAID [2]

Healthy men and women aged 18-70 years in general good health who were taking NSAIDs for at least three months because of arthritis were compared with non-NSAID using controls. Regular use of histamine antagonists or proton pump inhibitors was permitted, as was low dose aspirin.

After an eight-hour fast, the capsule was swallowed. Two independent blinded observers separately reviewed captured data for injury, from the point at which the capsule had passed the pylorus. Categories of injury were normal, red spots, small erosions, large erosions, or ulcers, using prior definitions.

Results

There were 21 NSAID users and 20 controls, predominantly men, and with an average age of 50 years. Two control subjects had signs of mild injury (Table 1). Fifteen NSAID users had signs of injury (71%) and in five (24%) the injury was severe.

Table 1: Small bowel lesions seen by video capsule endoscopy in 21 NSAID users and 20 controls not using NSAIDs. Use of H2A and PPI permitted

	NSAID users	Controls
Red spots	2	1
Erosions	8	1
Large erosion/ulcer	5	0
Total (%)	15 (71%)	2 (10%)

NSAID plus omeprazole [3]

Here 40 healthy volunteers not taking aspirin or NSAIDs initially underwent a faecal calprotectin test and video endoscopy. They then took 150 mg slow-release diclofenac and 40 mg omeprazole daily for two weeks, when calprotectin test and video endoscopy were repeated. Images were independently reviewed by three gastroenterologists blinded to identity and treatment. In this case the damage was scored as reddened folds with erythema, denuded area with loss of villous architecture, red spots, mucosal breaks, blood without visualised lesion, other lesions.

Results

Volunteers were 23 men and 17 women aged 21-60 years. They had no pathology initially, but 27 (68%) had some pathology after taking diclofenac 150 mg and omeprazole, with 15 (38%) having more than one category of lesion. Mucosal breaks were seen in 16, and blood in three. Thirty (75%) had faecal calprotectin levels above the upper limit of normal when taking diclofenac plus omeprazole compared with five (13%) beforehand; 36 showed an increase in faecal calprotectin with diclofenac.

Randomised trial [4]

A properly randomised and blinded (triple dummy) trial examined the effects of placebo, celecoxib 400 mg daily, and naproxen 1000 mg daily plus omeprazole 20 mg daily in healthy volunteers. Volunteers aged 18-70 years and not taking NSAIDs or aspirin were given an initial capsule endoscopy, and those without mucosal breaks randomised. After two weeks of treatment the endoscopy was repeated. Review of the images locally and centrally was blinded, with images judged using preset categories, eight of which were relevant.

Results

The average age of volunteers was about 33 years, and about 40% were men. The numbers randomised with usable images were 113 on placebo, 115 on celecoxib, and 111 on naproxen/omeprazole.

Significantly more volunteers had a mucosal break with naproxen (55%) than celecoxib (16%), and both were significantly greater than placebo (7%; Figure 1). Not only

Figure 1: Average number of mucosal breaks seen per patient by video-capsule endoscopy

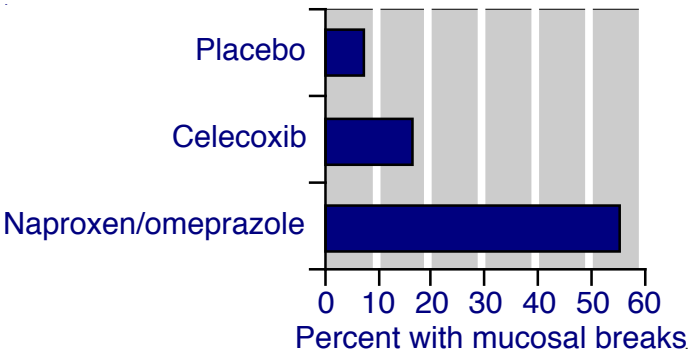


Table 2: Small bowel lesions in randomised comparison, using video capsule endoscopy

	Placebo	Celecoxib	Naproxen/ omeprazole
Small bowel mucosal breaks per patient	0.1	0.3	3.0
Any small bowel lesion per patient	0.1	0.5	4.2
Blood in small bowel, without visualised lesion (%)	1	7	8

were there more patients with small bowel breaks, but there were more breaks or lesions of any sort with naproxen/omeprazole than celecoxib, and again both were significantly greater than with placebo (Table 2). The number of occasions when blood was found without a lesion was the same for celecoxib and naproxen/omeprazole.

Comment

Capsule endoscopy studies are confirming what earlier studies have suggested, that NSAIDs are associated with a high prevalence of injury to the bowel. The clinical implication is that patients may bleed from their small intestine, and lose protein, and become anaemic. Anaemia is also associated with NSAID use, though studies are sparse so far.

Though the implication of each of the various lesions seen by gastroenterologists is less than clear (to Bandolier that is, not necessarily the gastroenterologists), the fact is that prevalence is high. It is also clear that proton pump inhibitors do not reduce this prevalence, and probably have little effect beyond the stomach. Celecoxib, and probably other coxibs as well, reduce the problem very substantially, perhaps by 80-90%, but do not eliminate it altogether.

Bandolier worries about anaemia as another problem with NSAIDs. A recent randomised trial comparing topical with oral diclofenac [5] showed that 10% of patients with knee arthritis on oral diclofenac developed anaemia in 12 weeks, much more than with topical diclofenac. There may be unresolved problems with anaemia in older people (Bandolier 137), who are much more likely to have arthritis and take NSAIDs. Much food for thought here.

References:

1 JA Tibble et al. High prevalence of NSAID enteropathy as shown by a simple faecal test. Gut 1999 45: 363-366.
2 DY Graham et al. Visible small-intestinal mucosal injury in chronic NSAID users. Clinical Gastroenterology and Hepatology 2005 3: 55-59.
3 L Maiden et al. A quantitative analysis of NSAID-induced small bowel pathology by capsule endoscopy. Gastroenterology 2005 128: 1172-1178.
4 JL Goldstein et al. Video capsule endoscopy to prospectively assess small bowel injury with celecoxib, naproxen plus omeprazole, and placebo. Clinical Gastroenterology and Hepatology 2005 3: 133-141.
5 PS Tugwell et al. Equivalence study of a topical diclofenac solution (PENNSAID) compared with oral diclofenac in the symptomatic treatment of osteoarthritis of the knee: a randomized controlled study. Journal of Rheumatology 2004 31: 2002-2012.

ISOTRETINOIN AND SUICIDE

Bandolier, like many of its readers, finds rare associations between an intervention and some rare but serious adverse event both interesting and challenging. It is interesting because any evidence that informs on how to avoid the adverse event is important. It is challenging because when adverse events are rare, determining causation or even rate is often difficult to the point of impossibility.

An example might be the use of isotretinoin for acne and reports of suicide, especially in young men. A search found three recent papers, two of them systematic reviews, which help to some extent, but will be deeply unsatisfying to anyone who wants to find a connection between treatment and suicide.

Systematic reviews

Both systematic reviews [1,2] searched three of four large and relevant databases, essentially for any study including the drug, and depression or suicide, and in humans. The formal studies could be cohort studies or randomised trials. One included case reports [1], while the other [2] did not.

Results

Surprisingly, given the general similarity in inclusion criteria, only four studies were common to both reviews.

The first review included case reports as well as formal studies, mainly from reports to registration authorities. Case reports included depression, psychiatric reactions, and some suicide ideation, suicide attempts, and completed suicides. In 25 cases documented by the FDA, cessation of drug was associated with resolution of mood disturbance, and reinstitution followed by a period of depression.

Many of the studies were small, and only two examined suicidal behaviour and isotretinoin use. One found no events, and the other, looking at attempted and completed suicide together, found 37 such events in 35,000 person-years of isotretinoin exposure. The only predictor was a previous history of depression or psychosis, which had an 8-fold increased risk.

New cohort study [3]

In 2003 all cases of psychosis associated with exposure to isotretinoin among conscripts in the Israeli Defence Force were reviewed. In this period 500 conscripts were seen for

severe acne by a dermatologist. There were five cases, three female, aged 19 or 20 years, all treated with isotretinoin before developing psychiatric morbidity. All had undergone pre-intake assessment at age 17, including a review of psychiatric history. The lag time was between three and 11 months, with a median of eight months. Three of the five soldiers had attempted suicide.

In all five cases there were predisposing factors, including obsessive-compulsive disorder, family history of schizophrenia or bipolar disorder, pituitary tumour, or intractable headache after head trauma. Three had more than one predisposing condition. One of the cases has a sibling with a manic episode after isotretinoin treatment.

Comment

It is impossible to say from the evidence available that there is a link between isotretinoin and suicidal behaviour. Neither is it impossible to say that there is no such link. But there are now two tenuous pieces of evidence to suggest that giving isotretinoin to young people with acne who either themselves have depression or psychiatric history, or who have a family history, may not be the best of ideas.

Young people have higher suicide risk, and acne has itself been associated with increased depression and suicidal behaviour. So is mental illness, and the lifetime risk of suicide is 1 in 20 in schizophrenia [4]. Very high suicide risks can occur on admission to and after discharge from psychiatric hospitals [5].

For a young person with acne and a personal or family history of depression or mental illness, the risk of suicide or suicidal behaviour must be high. Whether treating acne with isotretinoin increases the risk further is an almost impossible question to answer. It may be best avoided in those circumstances by appropriate history-taking.

The issue here, like so many other cases, is not so much whether therapies are effective or harmful, but in whom they are effective or harmful. This is a much under-researched area, where regulation and trials fail practice. It also asks some fundamental questions about our priorities.

References:

1 P Magin et al. Isotretinoin, depression and suicide: a review of the evidence. British Journal of General Practice 2005 55: 134-138.
2 AL Marqueling, LT Zane. Depression and suicidal behaviour in acne patients treated with isotretinoin: a systematic review. Seminars in Cutaneous Medicine and Surgery 2005 24: 92-102.
3 Y Barak et al. Active psychosis following Accutane (Isotretinoin) treatment. International Clinical Psychopharmacology 2005 20: 39-41.
4 BA Palmer et al. The lifetime risk of suicide in schizophrenia. Archives of General Psychiatry 2005 62: 247-252.
5 P Qin, M Nordentoft. Suicide risk in relation to psychiatric hospitalization. Archives of General Psychiatry 2005 62: 427-432.

Improving care for chronic illness

Chronic illness is common. Depending where you go to access statistics, it is as many as a third to a half of people in the USA, 90 to 125 million people with at least one chronic illness, with a fifth (50 million in the USA) with two or more chronic illnesses. Chronic illness is very expensive, and asthma, diabetes, heart disease, hypertension, and mood disorders account for up to half of all healthcare expenditures in more developed countries. Use of healthcare resources by someone with a chronic illness can be five times higher than by a healthy person.

Much chronic illness is in the community, and delivering the right care to the right patient in the right way could have significant benefits. In the USA a Chronic Care Model (CCM) has been developed as a framework for improving chronic disease care in the community. The CCM has six elements for providing high quality services (Table 1), four of which are directly related to care delivery. The question is whether these elements do contribute to better care, and a meta-analysis [1] suggests that they do.

Meta-analysis

Four clinical areas were of interest: asthma, congestive heart failure, depression, and diabetes. Systematic reviews and meta-analyses of chronic illnesses and electronic searches were used to find relevant studies. Included studies could be randomised or observational, as long as the intervention used at least one CCM element. Outcomes chosen were continuous or dichotomous clinical variables, some pre-

Table 1: Elements of the Chronic Care Model

General	Specific
Delivery system design	Care management roles
	Team practice
	Care delivery coordination
	Proactive follow-up
	Planned visit
	Visit system change
Self-management support	Patient education
	Patient psychosocial support
	Self-management assessment
	Self-management resources
	Collaborative decision-making
	Guidelines available to patients
Decision support	Institutional guidelines or prompts
	Provider education
	Expert consultation support
Clinical information systems	Patient registry system
	User information for care management
	Feedback of performance data
Community resources	For patients
	For community
Health care organisation	Leadership support
	Provider participation
	Coherent system improvement and spread

specified, like emergency department visits for asthma or congestive heart failure, or HbA_{1c} for diabetes. Quality of life information was also sought, and process of care indicators, like number of patients receiving appropriate medicines, or being tested for HbA_{1c}. Pooled estimates for interventions versus controls were then calculated in each of these areas, for each of the clinical conditions and overall.

Results

They found 112 studies, 93% of which were randomised. Most were published since 1999, so were recent. Most (107) were in an outpatient setting looking at one or two interventions, and half had follow ups of six months or longer. Of the randomised trials, few had good quality scores, but were adequate given that blinding was next to impossible. Only a third had reasonably large numbers (>200) patients.

Results are summarised in Table 2. For most of the outcomes over most of the conditions, use of a care package with at least one of the CCM interventions delivered a significantly better result. For instance, for diabetes this was equivalent to a 0.3% to 0.5% reduction in HbA_{1C}. Results were even better when restricted to randomised trials with better quality scores. Most information was available for interventions that included elements of delivery system design, decision support, or self-management support, and subgroup analyses for these were generally significant.

Comment

This all sounds a bit management-speak, and, to some extent, it is. A word of caution also, because none of the supporting links from the paper worked on the day that Bandolier tried. But this should not be dismissed. Material on the Internet shows that some primary care settings in the USA have had terrific results, improving patient care while reducing costs – what every decent care pathway should deliver. And delivery of healthcare is a complex business, not much studied. Perhaps we suffer from too much policy-based evidence, rather than evidence-based policy. Certainly this is worth a detailed look for those working in chronic care delivery.

Reference:

1 AC Tsai et al. A meta-analysis of interventions to improve care for chronic illness. American Journal of Managed Care 2005 11: 478-488.

Table 2: Analysis by element and overall, shaded areas showing statistical improvement

	Outcome variable			
Condition	Continuous	Dichotomous	Quality of life	Process of care
Asthma		9	12	2
CHF		19	6	6
Depression	27	14	3	15
Diabetes	25	4	3	9
Overall	52	46	24	32

Book review

Ceri J Phillips. Health economics: an introduction for health professionals. Blackwell Publishing, BMJ Books. ISBN 0-7279-1849-4. 150 pp; Cost £24.95 (November 2005)

The best present is the one you didn't know you wanted, but when you have it you wonder how you could have done without it. This is how this book feels.

It is easy to say what it is not. It is not a textbook; no reader will skip off with a happy heart and churn out health economic analyses and book a flight to Stockholm or Oslo. It is not the answer to the world, the universe, and everything. It is not the health economic equivalent of Caesar's response to Cato, an anti-NICE.

Rather it is a gently philosophical, sometimes witty, and always erudite opening into the arcane thinking of health economics for the general reader. It takes a broad view of the importance of economic thinking in the running of health services, and specifically in the running of the National Health Service in the UK. It does not hide in the dark corners of cost-effectiveness, but asks searching questions about how to get the mostest with the leastest, in the bestest way.

Is there an answer? Well, yes and no. It is clearly possible to do better, and Phillips points out some of the incongruities that follow complex budget-based decision-making rather than taking a more holistic view. But there is no getting off the hook of responsibility, personal and collective. In his conclusion, he quotes Chesterton's response to a Times quest for essays on what was wrong with the world: "I am".

Do a good deed for yourself or someone else: a present for the new year. First, you will enjoy the read, because it is well written. Second, you will know just enough more about health economics to feel informed. Third, you will be able to use that knowledge to ask searching questions about what you and others do and how you do it. Fourth, it will probably send some of you looking for ways to accomplish that.

But this is a book not just for the professional. It is accessible for any reasonably informed reader. Particularly in the UK where so many major changes are taking place in healthcare, it will make you ponder whether this change is the best, in the best of all possible worlds.

EDITORS

Andrew Moore Henry McQuay
Pain Relief Unit
The Churchill, Oxford OX3 7LJ
Editorial office: 01865 226132
Editorial fax: 01865 226978
Email: andrew.moore@pru.ox.ac.uk
Internet: www.ebandolier.com
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